Testimony of

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On the

Legislative Hearing on

S. 742 and Draft Legislation to

Ban Asbestos in Products

Subcommittee on

Environment

And Hazardous Materials

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Summary

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I am writing to share my views concerning the legislation proposing to ban asbestos in America. I am very familiar with the health effects associated with the inhalation of asbestos having been born in Paterson, NJ, where an asbestos factory produced amosite asbestos insulation for US Navy ships in the Second World War. As I was growing-up in the 1960s sufficient time had past for some of my neighbors (who had worked in this factory decades earlier) to develop asbestos-related diseases. More than twice as many people died from asbestos-related diseases in Paterson, NJ, than in Libby, Montana.

I took a serious interest in trying to understand how and why this happened. I joined Dr. Irving J. Selikoff's research group at Mount Sinai School of Medicine in New York City. At the time, he was a world leader in asbestos research and a long time resident of New Jersey. Also, I shared his opinion that a ban of asbestos was not necessary and it is an old

idea which has been seriously considered and rejected when it was found not to be supported by the facts. I have a doctoral degree in chemistry and a member of the faculty in both Chemistry and Earth and Environmental Sciences of the The Graduate School and University Center of the City University of New York.

Definition of Asbestos and Analytical Method for Assessing Exposure

The definitions of asbestos used in the legislation to ban asbestos are not specific enough (as written S.742) to apply only to asbestos and therefore the ban would include other non-asbestos fibers. These non-asbestos fibers are described as being "elongated mineral particles" and "biopersistent" occur very commonly in nature and should not be included in an asbestos ban. The U.S. Occupational Safety and Health Administration (OSHA) does not regulate these non- asbestos fibers after having a rulemaking to determined they do not present health hazards similar to asbestos. I would recommend the deletion from the legislation of the "elongated mineral particles" and "biopersistence" so the ban specifically addresses the commercial asbestos minerals.

Three other minerals are referred to by name in S.742 to be included in the ban, none of which has ever been regulated as asbestos – richterite, winchite, and erionite. The International Agency for Research on Cancer (IARC) has determined fibrous erionite is a human carcinogen (Group 1), but there has never been an erionite-related mesothelioma reported in the United States. The two other "durable fibers" mentioned are richterite and winchite, neither of which is classified as Group 1 by IARC. It has been known for decades that these minerals are present in the vermiculite deposit at Libby, Montana. Neither of these two fiber-types has ever been regulated as asbestos by OSHA. The predominant fiber in Libby is tremolite-actinolite asbestos. If these other three minerals

are to be including in the ban they should be referred to as “asbestiform erionite”, “asbestiform richterite”, and “asbestiform winchite”.

The ban asbestos legislation calls for banning minerals or products, which contain asbestos “in any concentration”. Modern analytical methods can identify extremely low concentrations of mineral fiber present in ore deposits, which may or may not be asbestos. The health effects of asbestos have historically been controlled by monitoring the concentration of airborne fibers, assuming all health effects arise from the inhalation of the asbestos.

There is no generally accepted method of predicting airborne fiber levels from the concentration of asbestos in an ore body. This approach becomes even move problematic as the fiber concentration in the bulk material decrease to 0.25% as described in the Committee Print for the aggregate products. The asbestos ban legislation as written may cause the presence of asbestos at low concentrations to be claimed where it is not present (Langer *et al*. 1991). I would recommend that the concentration of airborne fiber levels be used to monitor the workplaces where aggregate is produced, transported, and used. The Committee Print should eliminate the request for the Administrator to develop an “Asbestos Test Method” and not set a limit on the asbestos content of aggregate.

It is important to note that US consumption of aggregate is approximately 3 billion tons per annum and the suggestion that sampling methods be established to determine this amount of rock is less than 0.25% asbestos is not a scientifically justified approach to this problem. What is of interest is the exposures associated with the life cycle of this product. Particularly in the aggregate industry which has a long history of production with no suggestion of increased risk of asbestos-related disease.

An asbestos ban will not address the issues related to asbestos outcropping in areas of the country (often incorrectly referred to as "naturally occurring asbestos") or stop the amphibole asbestos exposures in Libby, Montana. The EPA Inspector General has noticed that EPA has not planned or completed a risk and toxicity assessment for the amphibole asbestos exposures in Libby, Montana, to determine the safe human exposure. The remediation measures in Libby taken to date are not based on a health standard which is the same comment another Inspector General's report made about the air sampling in the area of the World Trade Center post-9/11 (Office of the Inspector General 2006, Office of the Inspector General 2003, Nolan *et al.* 2005). The Ban Asbestos Bill would make managing these types of asbestos exposures (which cannot be eliminated by an asbestos ban) more difficult by falsely claiming no safe level exists.

History of the Asbestos Ban in the United States

The federal government's effort to ban asbestos started with an advanced notice of proposed rulemaking by the US Environmental Protection Agency on October 17, 1979. That year the total US consumption of asbestos was 560,000 tons and about 6.6% was the very carcinogenic amosite asbestos and crocidolite asbestos the balance being the less potent chrysotile asbestos (Hodgson and Darnton 2000). A year prior to the asbestos ban being proposed the National Cancer Institute and the National Institute of Environmental Health Sciences predicted that 2 million premature cancer deaths would occur over the next thirty years from past asbestos exposure or "roughly 17% of the total cancer incidence experienced in that period" (Efron, 1984). This prediction was based on the assumption that *any worker exposed to any type or concentration of asbestos* would have an asbestos-related cancer risk similar to an asbestos insulation workers. This

assumption is incorrect and the predicted numbers of asbestos-related cancer deaths have not occurred but it did drive the regulatory climate at the time leading to a call for an asbestos ban.

On October 18, 1991 the 5th Circuit Court of Appeal vacated EPA's ban because the agency had "failed to muster substantial evidence" to support the rule In 1986 the EPA estimated that a ban on asbestos shingles would "cost $23-34 million to save 0.32 statistic lives ($72-106 million per life)." The 5th Circuit went on to query why EPA would consider asbestos so dangerous if for example "...over the next 13 years, we can expect more than a dozen deaths from ingested *toothpicks*-a death toll more than twice what the EPA predicts will flow from the quarter billion-dollar bans on asbestos pipe, shingles and roof coatings."

The Court of Appeal's decision remanded the matter back to EPA to muster further evidence to support their claim that asbestos exposure constitutes an "unreasonable risk". EPA never provided such additional evidence and many would argue that for chrysotile asbestos it does not exist. The ban asbestos legislation has not addressed any of the Court of Appeal's concerns about mustering substantial evidence. Controlled use of chrysotile asbestos is feasible and it is happening in many parts of the world (Nolan *et al.* 2001). From time to time there are calls for a ban on asbestos but the "substantial evidence" the 5th Circuit asked for to show that controlled asbestos exposure presents an "unreasonable risk" is not available and arguments have been offered that such evidence does not exist (Wilson *et al.* 2001).

Asbestos Exposures in the US have Changed

In the 1970s when all the commercial asbestos fiber-types were being used in the United States, asbestos consumption was above 500,000 ton per year and the permissible asbestos exposure level (PEL) was 12 fibers/ml (equal to or great than 5 microns in length) an asbestos ban may have been justifiable. Since the U.S. Occupational Safety and Health Administration began to regulate asbestos in 1971, we would like to point out the events that have occurred to eliminate any scientific justification for an asbestos ban in the United States.

The permissible exposure limit (PEL) has been reduced to 0.1fibers/ml or 120-fold lower than the 1971 asbestos standard and hundreds of times lower than the historical high asbestos exposure levels associated with asbestos-related disease (Figure 1). The current US permissible exposure level for asbestos is among the lowest in the world. The statements that appear in the Ban Asbestos Bill indicating the current US permissible exposure level is not safe are not supported by reference to the medical and scientific literature and I would argue that such support does not exist (Nolan *et al.* 2001).

Asbestos-related disease in the United States can be divided into three different time periods: a historically high exposure period that resulted from poorly controlled use of asbestos from which our knowledge of the asbestos-related diseases is derived. This historical period ended with the Occupational Safety and Health Administration (OSHA) promulgating an asbestos permissible exposure limiting (PEL) in 1971, next followed a transition period when occupational asbestos exposures were lowered. The transition period ended in 1994 when the current 0.1f/mL permissible exposure limit for asbestos was adopted beginning the modern period of controlled asbestos use (Figure 1).

The latency period (from first asbestos exposure during the historical period to the development of asbestos-related cancer) *is* at least 15 years and in epidemiology studies it is generally 20 to 25 years before significant increases in asbestos-related cancers occur. Most, if not all, of the asbestos epidemiology studies analyzed the health outcomes of workers exposed to asbestos before 1971.

In their testimony before the Senate, NIOSH indicated a continuing interest in asbestos-related disease among a cohort of South Carolina textile workers. The plant opened in 1909 and closed their doors in 1977. The type of asbestos products manufactured in South Carolina are no longer produced or used in the US. Among the 1,841 deaths in the cohort (about 65% of the total workforce), there were three mesothelioma or 0.16% is the same as the general male US population (0.17% see Table 1). One would expect it to be higher due to their chrysotile asbestos exposure.

As a group, the three major US chrysotile exposed cohorts have reported 2,002 deaths with three mesotheliomas (Table 2). The risk of mesothelioma among the males in these three chrysotile-exposed cohorts is less than that experienced by the general males population (Table 1 & 2). In the US one male in 600 dies of a mesothelioma or 0.17% while in the chrysotile exposed cohorts three mesotheliomas occurred in 2,002 deaths or 0.15%. The Relative Risk (RR) is 0.88 (Table 2). Higher RRs for mesothelioma is associated with chrysotile mining and milling where exposures were significantly higher (Table 2).

What has changed to justify the US legislation to ban asbestos now?

Now as we re-visit the ban issue almost 16 years later much has happened to make a complete ban of asbestos in the US an even less attractive public health policy. In 1992 the dangerous amosite asbestos left commerce worldwide to be followed by crocidolite asbestos in 1997 (Figure 2). Crocidolite is the fiber-type first associated with mesothelioma in South Africa. This fiber-type is particularly potent and can cause mesothelioma after low exposure; this observation began the public health concern about non-occupational exposure to asbestos causing cancer (Table 3). Crocidolite asbestos and high exposure to amosite asbestos are the major etiological agents in this disease. Consumption of these two amphibole asbestos fiber-types started to decline in the 1960s and the US incidence of mesothelioma has been declining since the 1990s (Weill *et al.* 2004). These favorable trends are not commonly known or appreciated.

US consumption of asbestos has fallen to 1,500 tons of chrysotile asbestos in 2007 which is less than ¼ % of the consumption in the mid-1970s (Figure 2). Exposures are much better controlled. Most of the chrysotile asbestos the legislation would ban is used in asphalt roofing products that are not regulated by the U.S. Occupational Safety and Health Administration as an asbestos-containing product because there is no evidence of asbestos release from this matrix.

Less than 17% of the countries around the world have chosen to ban asbestos (most after the EPA ban was vacated in 1991) but worldwide consumption has remained in excess of 2,000,000 tons per annum. Most of the asbestos bans were not total but were to ban certain uses of asbestos while other critical uses such as gaskets to contain

corrosive gases, in rocket engines and diaphragms for production of chlorine are allowed. The US Court of Appeal review is unique in that openness of the US Judicial process allowed for an impartial review of a government led asbestos ban. Such an open review of government policy simply does not happen in other countries. To our knowledge the issues raised by the 5th Circuit have never been addressed in any country where asbestos has been banned.

The significantly higher carcinogenic potency of the commercial amphibole asbestos minerals (amosite and crocidolite) compared to chrysotile has been well understood for a many years with the latest quantitative risk assessment by Hodgson and Darnton appearing in 2000 (Table 3). The most recent estimate in the range of potency between crocidolite asbestos and chrysotile asbestos for mesothelioma is 500 to 1. These are large differences and offer an explanation why a single occupational exposure standard based on averaging would not yield an effective permissible exposure standard. More recently after reviewing the epidemiology available for assessment of chrysotile asbestos as a cause of mesothelioma Yarborough 2006 concluded that the "risk of chrysotile for mesothelioma in most regulatory context reflects public policies, not the application of the scientific method as applied to epidemiology studies." Yarborough is not supporting the claim in S.742 that the current asbestos permissible exposure limit does not protect workers.

Historical Legacy from High Exposure and Amphibole Asbestos

The first asbestos exposures in all the other major asbestos cohort studies also began decades prior to the beginning of the transition period in 1971. Due to the long latency for asbestos-related cancers, cases continue to develop from these exposures. Projections

indicate that new cases will continue, in decreasing numbers, until about 2055. These cases are the historic legacy from poorly controlled use of asbestos which some have referred to as the iron grip of latency. Those exposures occurred many years ago and their consequences *cannot be undone* by any legislation or public health action.

Currently, the US is using around 1,500 tons of chrysotile asbestos a year to fabricate a very limited number of asbestos-containing products that release little or no fibers (asphalt roofing, chlorine gas processing, and insulation for the space shuttle). Others and S.742 have tried to justify an asbestos ban by claiming the US is using unlabeled asbestos products; I find no evidence for this and consider their claim false. OSHA and CPSC requires most asbestos-containing products to be labeled as such and OSHA requires exposures since 1994 to be controlled at the PEL of 0.1f/mL.

About 14 years have passed since the current asbestos standard was adopted. Therefore, the latency period is insufficient to observe the mortality experience of modern asbestos workers. Asbestos workers who from high exposures developed asbestos-related cancers were used to parameterize the risk assessment. To understand the trend of asbestos-related disease in the modern period I will rely on a risk assessment and predict the number of future asbestos-related cancer at current exposure level.

What are the Risks of Asbestos-Related Cancer with the Controlled Use of Chrysotile Asbestos?

This question can be answered by doing a risk assessment for asbestos-related cancer.

This risk assessment, which is simple, straightforward and follows principles well established in the last 20 years, should be addressed first. Then, if these risks are unacceptable, you should proceed with banning asbestos. If, on the other hand, the risks are negligible, the time of the people and the Congress should not be wasted. There are already too many unnecessary laws. The cohorts of asbestos exposed workers used to develop the risk assessment were exposed primarily, if not exclusively, prior to 1971 and therefore represent the historic period of asbestos exposure and will be used to predict the future.

I will show that this is an excellent example of how we have learned from our past mistakes and I will argue that we have already taken all the action necessary to avoid a repetition or continuation of hazard from asbestos-related cancer.

Exposure to 0.1f/mL, the PEL since 1994, for 45 years leads to a cumulative exposure of 4.5f/mL x years. In their Senate testimony on June 12, 2007, NIOSH claimed asbestos exposure at the current PEL would cause 3.4 asbestos-related cancers per 1,000 workers over their lifetimes. The NIOSH projection is based on a risk model developed in 1986 that projects average risk for exposure to all three commercial asbestos fiber-types – crocidolite, amosite, and chrysotile used in commerce in the US prior to that time.

Currently only chrysotile asbestos continues to be used in commerce in the US and worldwide. Therefore, to project future asbestos-related cancers I used the asbestos risk assessment developed by Hodgson and Darnton in 2000, which projects cancer risks for the individual asbestos fiber-types. These epidemiologists work at the Heath Safety Executive, an agency of the United Kingdom government and work independently of

both labor and management influence. The overwhelming majority of the public believe asbestos to be a single substance not six different minerals.

These predictions differ from those provided by NIOSH in three important ways:

First, NIOSH's estimates are higher than those for chrysotile asbestos and lower than for the two commercial amphibole asbestos fiber-types. A similar asbestos exposure leads to a significantly lower health hazard for a chrysotile-exposed population than, for a population otherwise identical, exposed to commercial amphibole asbestos. I argue that NIOSH's choice to average the potency of the fiber-types is a significant contributing factor to any asbestos-related disease on workers starting during the transition period (Figure 1, Table 4).

Secondly, our asbestos-related cancer risk due to commercial amphiboles exposure is reported as a range while NIOSH has a single value. This is to mirror the different rates of the lung cancer from amphibole asbestos exposures occurring in the different cohorts of workers. Each cohort is exposed to only one of these two fiber-types. Lung cancer as a function of exposure is similar for the two amphibole asbestos fiber-types, between 10 to 50-fold greater than for chrysotile, NIOSH's prediction lacks this texture (Hodgson and Darnton, 2000).

Third, I assume the workers do not smoke while NIOSH did not say if they smoke or not. The lung cancer risk is 10-fold higher for smokers than those who do not smoke. Exposure to all types of asbestos increases your lung cancer risk as a proportion of your underlying lung cancer risk (Figure 3). Therefore, the lung cancer risk among smokers

for a given asbestos exposure would be greater than in a non-smoker. High cumulative exposures to asbestos can cause a dramatic increase in lung cancer while at low cumulative exposure the dominant risk factor is your smoking habit (Figure 3). With the modern PEL since 1994, the major risk factor for lung cancer is whether you smoke or not.

NIOSH opined that asbestos is the leading cause of lung cancer among non-smokers. It is not clear how they came to this conclusion. Lung cancer risk among non-smokers is about 8 lung cancer cases per 1,000 non-smokers deaths. The increase in the lung cancer risk due to asbestos is calculated as a proportion to the underling lung cancer risk with modern controlled exposures will always be very small about 0.022 lung cancer deaths for 1,000 asbestos workers from a lifetime of asbestos exposure at the modern PEL. Other causes of lung cancer besides tobacco are arsenic, mustard gas [chloromethyl (pyriline) ethers], polycyclic aromatic hydrocarbons, hexavalent forms of chromium, air pollution including fine particles, ionizing radiation and radon (Higginson *et al.*1992).

The fiber-type specific lifetime risk for chrysotile asbestos is more than 50-fold below the NIOSH prediction for exposure to fiber of average potency. NIOSH averages in the higher effects for the amphibole asbestos fiber-types and our calculation provides more texture as to the contribution of the different fiber-types.

At the Senate Hearing NIOSH stated that its goal is to have an upper limit of 1 occupationally-related death per 1,000 worker lifetimes and NIOSH went on to claim that asbestos at the current PEL does not meet that goal (it is 3.4 per 1,000). With the departure of commercial amphibole asbestos from commerce, chrysotile asbestos at the current PEL is about 15-fold below NIOSH's target goal (Table 4). If the asbestos-related

cancers associated with the more potent commercial amphibole asbestos fiber-types are excluded NIOSH's rationale for asbestos regulation falls to the ground.

The more significant risk associated with the amphibole asbestos has been removed from the US by market forces rather than the government taking regulatory action to do so. The proposed ban would not affect amphibole asbestos *currently in place or exposures that have already occurred.*

Predictions for the number of asbestos-related deaths presented at the Senate Hearings are all from exposures that occurred before the modern period. The asbestos-related diseases are currently occurring among individuals whose cumulative asbestos exposures are higher than allowed in the modern period and in many cases were to asbestos fiber-types, i.e. amphibole asbestos, no longer used.

Risk assessment is also useful for predicting the total number of asbestos-related deaths associated with a public health policy of controlled use of chrysotile asbestos.

In 2003 the total number of deaths in the United States was 2,448,250. If all of these people worked with chrysotile for 45 years at the current permissible exposure level of 0.1f/mL there would have been 164 asbestos-related cancer deaths based on our risk estimate for chrysotile of 0.067 asbestos-related cancers per 1,000 deaths.

I know of no one who would argue that the entire US population had such a significant asbestos exposure. So this number of asbestos-related deaths would not be realized.

I estimate at most 1% (24,483) of deaths would have such a high cumulative exposure based on the current US asbestos consumption and uses. It seems very unlikely that 24,483 people would be exposed to asbestos considering its current usage. Therefore, continuing with our policy of controlled use of chrysotile asbestos, once time eliminates the legacy of asbestos-related disease from past exposures, less than two asbestos-related deaths would be expected to occur each year in the United States. The 10,000 deaths in the S.742 are not going to occur from the current US asbestos permissible exposure limit.

As the 5th Circuit Court noted in their opinion striking down EPA's attempt to ban asbestos-containing products that some risks of asbestos-related cancer are similar to choking to death on a toothpick (United States, 1991).

I conclude that in the United States, all the steps required for mitigating the health effects associated with asbestos exposure have already been taken and a ban on chrysotile asbestos will be no more than a symbolic gesture and not have any practical effect.

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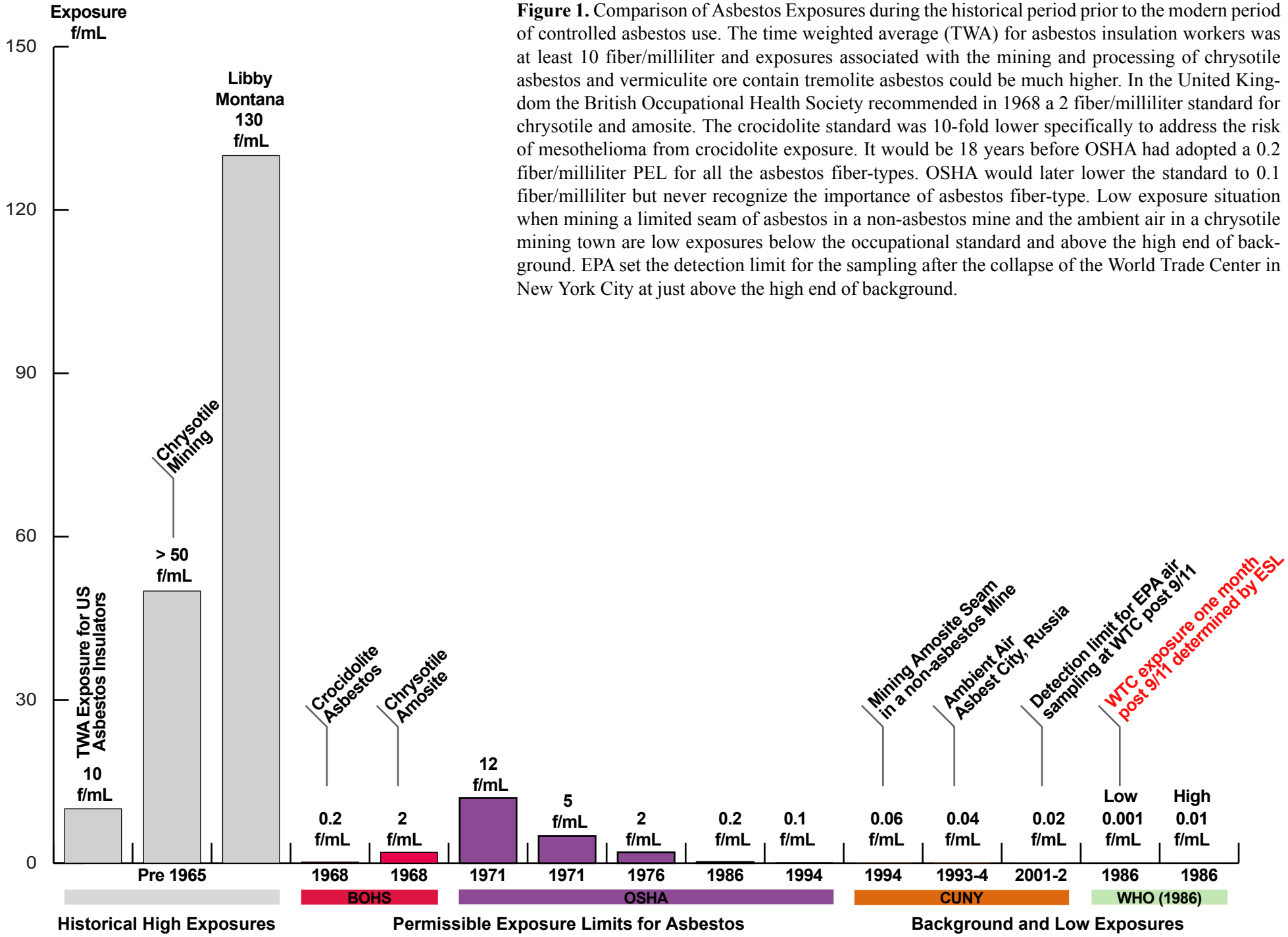

Figure 1. Comparison of Asbestos Exposures during the historical period prior to the modern period of controlled asbestos use. The time weighted average (TWA) for asbestos insulation workers was at least 10 fiber/milliliter and exposures associated with the mining and processing of chrysotile asbestos and vermiculite ore contain tremolite asbestos could be much higher. In the United Kingdom the British Occupational Health Society recommended in 1968 a 2 fiber/milliliter standard for chrysotile and amosite. The crocidolite standard was 10-fold lower specifically to address the risk of mesothelioma from crocidolite exposure. It would be 18 years before OSHA had adopted a 0.2 fiber/milliliter PEL for all the asbestos fiber-types. OSHA would later lower the standard to 0.1 fiber/milliliter but never recognize the importance of asbestos fiber-type. Low exposure situation when mining a limited seam of asbestos in a non-asbestos mine and the ambient air in a chrysotile mining town are low exposures below the occupational standard and above the high end of background. EPA set the detection limit for the sampling after the collapse of the World Trade Center in New York City at just above the high end of background.

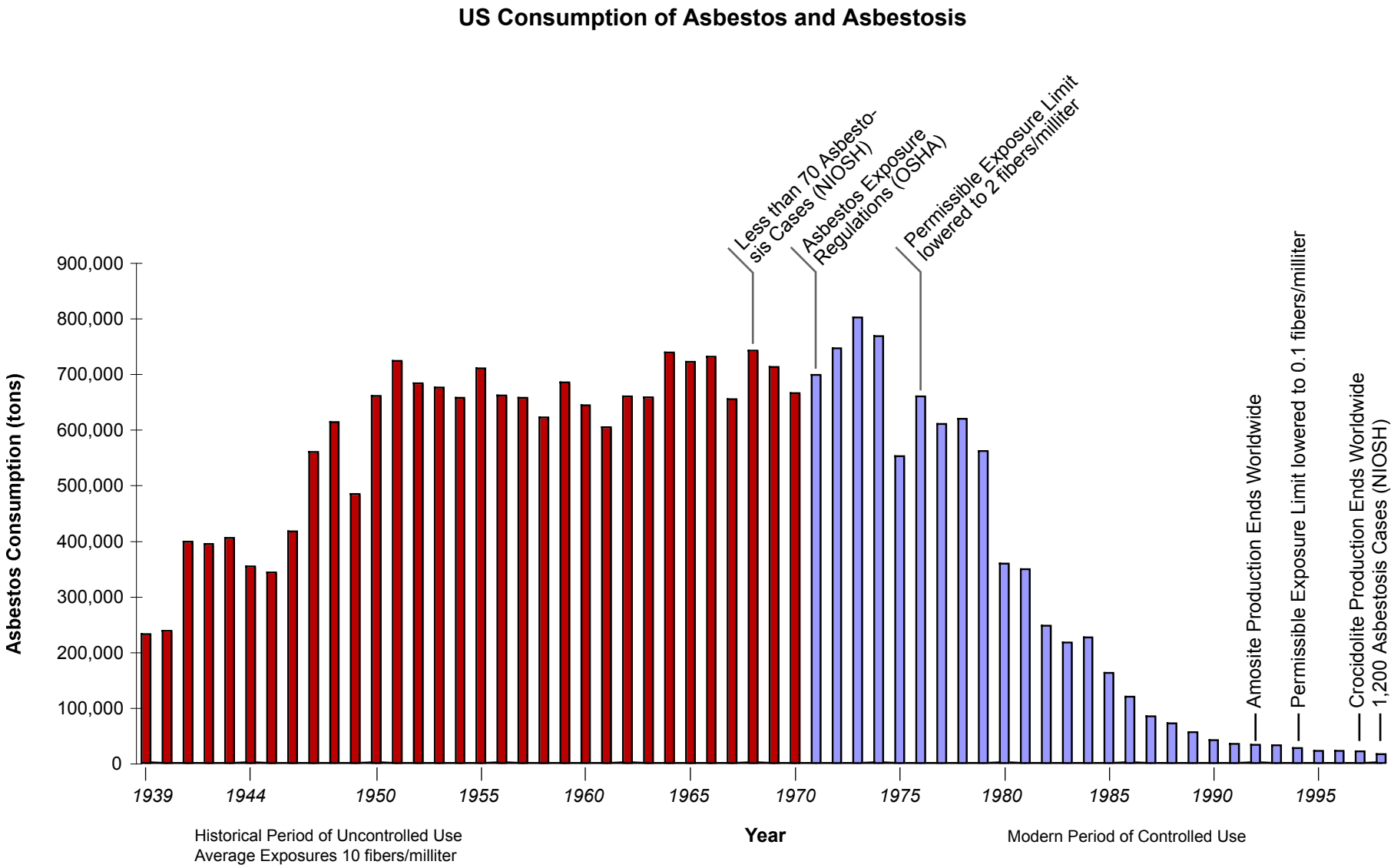

Figure 2. United States consumption of asbestos from 1939 to 1968 was 17,310,187 tons and airborne asbestos exposures were approximately 10 fibers/milliliter for Time Weighted Averages (certain task involved significantly higher exposures). From 1969 to 1998 US consumption of asbestos decreased to 9,523,469 ton and a permissible exposure limit for asbestos was adopted that was reduced to 0.1fiber/milliliter by 1994. Why did the number of asbestosis cases increase from less than 70 in 1968 to around 1,200 in 1998?

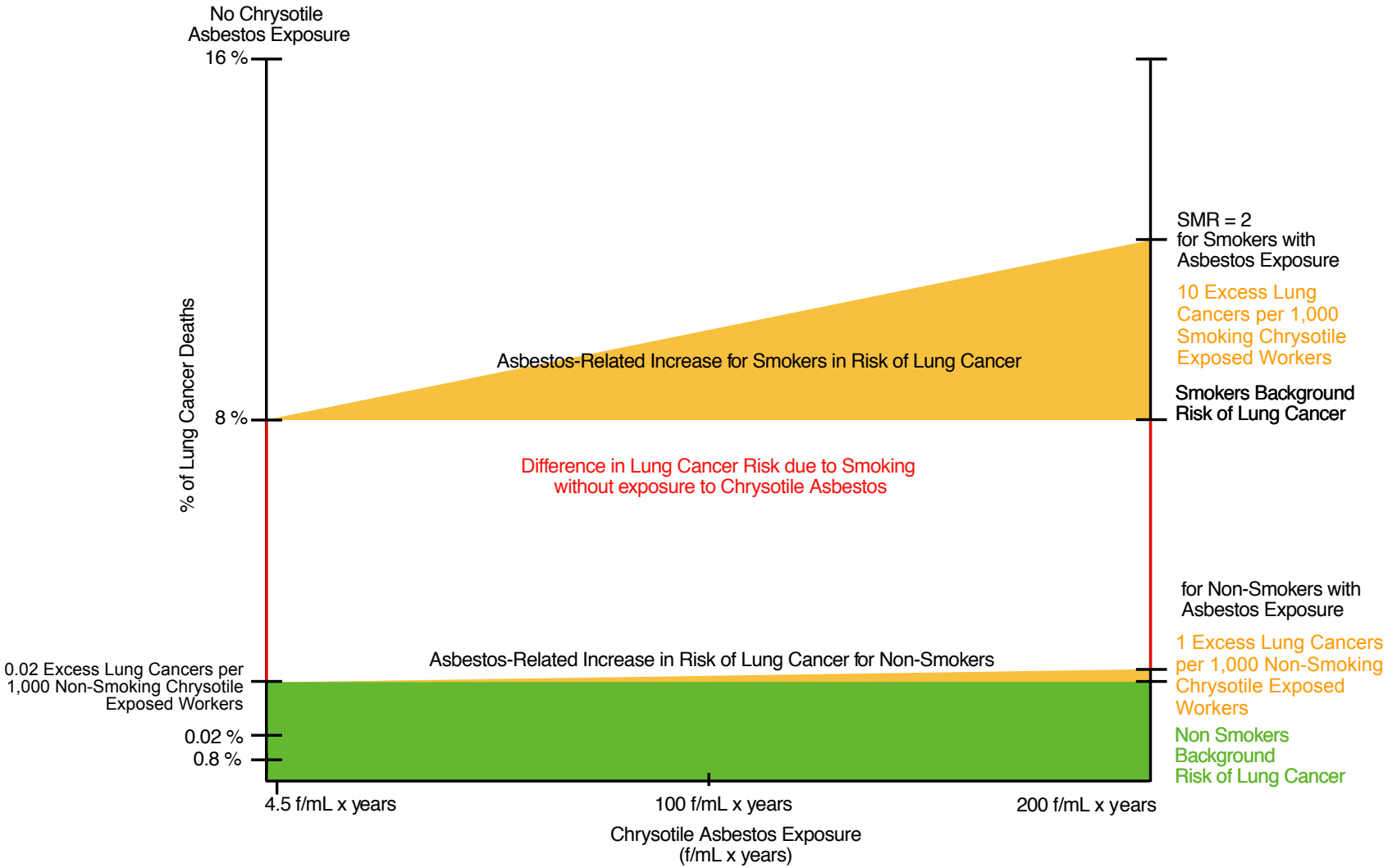

Figure 3. Exposure to asbestos increases lung cancer risk by 0.02 per 1,000 chrysotile asbestos workers with 45 years of exposure at the 0.1 fiber/milliliter asbestos permissible exposure limit. Smokers experience an excess lung cancer risk which is 10-fold greater. In their testimony NIOSH indicated 1 excess death over a working lifetime or less was the target. Approximately 200 fiber/milliliter x years of chrysotile exposure approach 1 excess asbestos-related lung cancer in 1,000 worker.

Table 1. The percentage of deaths due to mesothelioma in the United States in 2003 is given for the general population and the specifically males and females. About 2,560 mesotheliomas occurred in the United States in 2003 where the disease was about 4-fold more common in males than females. 2,448,288 deaths occurred in the US that year with 1,201,964 in males. US males in 2003 are expected to have 1 mesothelioma in 600 deaths. Recently in the US general population mesothelioma accounts for 1 death in 1, 000 deaths in the general population and 1 in 600 and 2,000 for males and females respectively.

Asbestos Fiber-Type	Mesothelioma Deaths Per 1,000 in General Population	Total № of Mesotheliomas/ Deaths (%) §	Average Cumulative Exposure f/ml x Years
United States	**1**	**2,560(0.1%)**	***Bkgd***†
Males	**1.7**	**2,000(0.17%)**	***Bkgd***
Females	**0.45**	**560(0.045%)**	***Bkgd***

†World Health Organization (1986) estimated the global background for asbestos in the ambient air to be between 0.001 and 0.01 fibers/milliliter and chrysotile is the predominant fiber-type.

§Mesothelioma as a percentage of all deaths.

Table 2. Data for the five chrysotile-exposed cohorts all the mesotheliomas are pleural. General causation for chrysotile asbestos exposure doubling the background risk of pleural mesothelioma is marginal among the Quebec miners and millers. It critically depends upon the estimate of background mesotheliomas. Adding in manufacturing workers makes the evidence weaker.

Fiber type	Name and Location	Mesotheliomas /all deaths (%)	Exposure f/ml x yrs	Risk Ratio Obs fraction /background
	Miners & Millers			
	Canadian Mines	33/7,456 (0.44%)	600	**2.0 (0.44/0.22)**
	Manufacturers			
Chrysotile	**Charleston South Carolina** *Males only in Charleston SC*	3/1,186 (0.25%) §	28	**1.5** (0.25/0.17)
Chrysotile	**New Orleans, LA**	0/259 (0%)	22	**0**
Chrysotile	**Connecticut**	0/557 (0%)	46	**0**
	All Males Manufacturers	***3/2,002 (0.15%)***		**0.88 (0.15/0.17)**
	TOTAL all studies	***39/10,540 (0.37%)***	***170***	**1.5**

§ Hein *et al.*2007 the other data are from Hodgson and Darnton, 2001.

Table 3. Mesothelioma mortality in 10 epidemiologic cohort studies of individuals exposed to crocidolite, amosite, actinolite asbestos and tremolite asbestos where general causation is well established. The average cumulative exposures are from Hodgson and Darnton, 2000 while the Risk Ratios (RR) have been added. Only the cohorts with occupational exposure to amphibole asbestos were useful for establishing causation. The non-occupational cohort studies were either negative or suggestive.

Asbestos Fiber-Type	Cohort Name	Total № of Mesotheliomas/ Deaths (%)	Mean Cumulative Exposure f/ml x Years§	Risk Ratio (>2)
Crocidolite	Miners			
	South Africa(SA)	20/423 (4.7%)	16.4	28(4.7/0.17)
	Wittenoom, Australia	72/719(9.1%)	23	54(9.1/0.17)
	Factory Workers			
	Massachusetts	5/28 (17.8%)	120	104(17.8/0.17)
Summary		***97/1,170(8.3%)***	***53***	**48 (8.3/0.17)**
Amosite	Paterson, NJ			
	Workers	17/740(2.3%)	65	13.5(2.3/0.17)
	Household	4/115 (3.5%)	Unknown	20.5(3.5/0.17)
	Neighborhood	1/780(0.12%)	Unknown	0.7(0.12/0.17)
	Tyler, TX	6/222(2.7%)		16(2.7/0.17)
	Uxbridge, UK	5/333(1.5%)		6(1.5/0.17)
	South African Miners	4/648(0.6%)	23.6	3.5(0.6/0.17)
Summary		***37/2,838(1.3%)***	***47.2***	**7.7**(1.3/0.17)
Tremolite-Actinolite Asbestos	Miners, Libby, MT†	**12/286 (4.2%)**		**25**(4.2/0.17)
Mean for four amphibole asbestos minerals		**146/4,294 (3.4%)**	***50***	**20(3.4/0.17)**

§Hodgson and Darnton, 2001. † McDonald *et al.* 2004.

Table 4. The lifetime risk from both asbestos-related cancers (lung and mesothelioma) are totaled and shown as the lifetime risk at the current PEL. At the Senate Hearing, NIOSH claimed 3.5 asbestos-related cancers per 1,000 for their asbestos fiber with average potency and opined the target was to get below 1 per 1,000. Note that for chrysotile the lifetime cumulative exposure at the current permissible exposure is about 15-fold below this while the commercial amphibole asbestos fiber-types are between 4.7 and 23.6-fold above the target. NIOSH needs to include the cancer risks associated with the amphibole asbestos fiber-types otherwise their regulatory policy falls to the ground.

Source	Asbestos Fiber-Type	Cumulative Asbestos Exposure	Lifetime Risk of Asbestos-Related Cancer (per 1,000 deaths)
NIOSH	Average for Mixed	4.5 f/mL x years	3.5†
NIOSH-Target			<1
Hodgson & Darnton	Chrysotile	4.5 f/mL x years	0.067(67%)§
Hodgson & Darnton	Amosite	4.5 f/mL x years	4.7-5.6(96%)
Hodgson & Darnton	Crocidolite	4.5 f/mL x years	22.7-23.6(99%)

§Percentage of risk associated with mesothelioma.

† NIOSH estimates are for asbestos-related lung cancer, mesothelioma, and gastrointestinal cancer. I limit this analysis to lung cancer and mesothelioma. Recently the Institute of Medicine concluded that among other cancers historically associated with asbestos a casual relationship is likely only for laryngeal cancer (IOM, 2006). There is no asbestos risk assessment for laryngeal cancer. However, 80-90% of laryngeal cancers are related to alcohol and smoking (Higginson *et al.* 1992), therefore asbestos-related laryngeal cancers are not included in our projections. It is rare disease in women and about 10-fold less common in males than smoking-related lung cancer (Higginson *et al.* 1992).